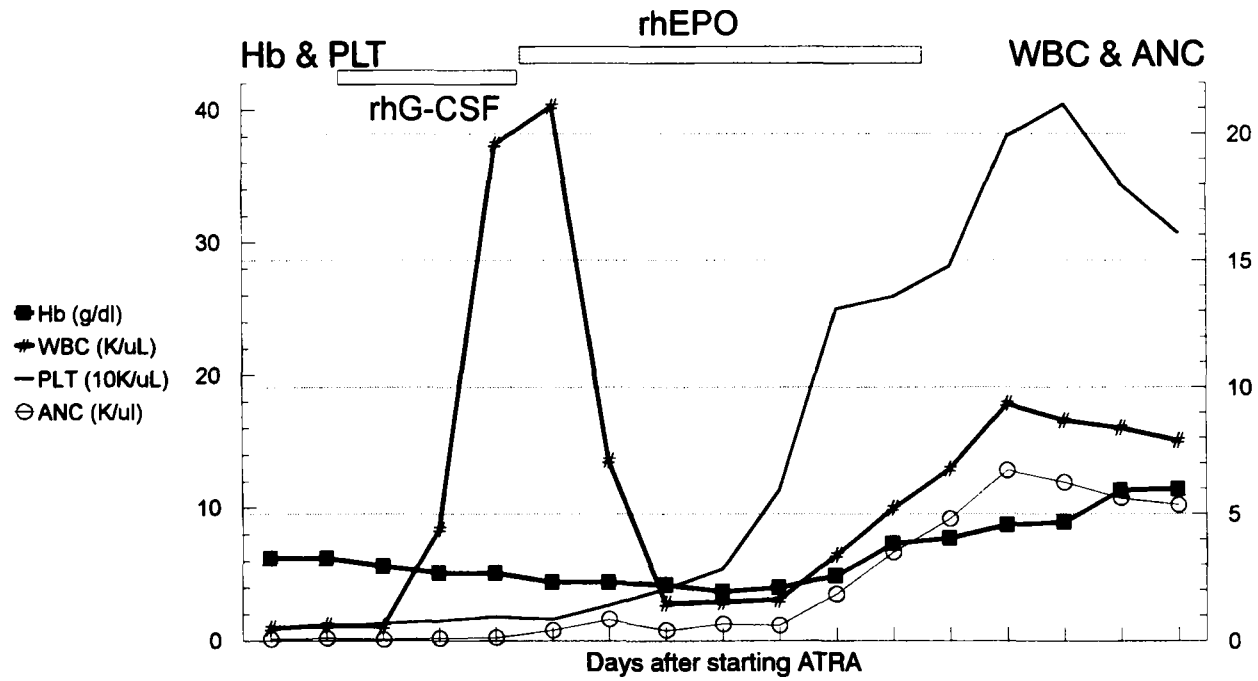

	1	3	5	7	9	11	14	18	22	25	30	36	43	54	59	75	96
Hb (g/dl)	6.2	6.2	5.6	5.1	5.1	4.4	4.4	4.2	3.7	4	4.9	7.3	7.7	8.7	8.9	11.3	11.4
WBC (K/uL)	0.5	0.61	0.58	4.4	19.59	21.08	7.11	1.46	1.53	1.61	3.38	5.21	6.76	9.33	8.67	8.39	7.88
PLT (10K/uL)	1.1	1	1.3	1.5	1.8	1.6	2.7	3.9	5.4	11.3	25	25.9	28.2	38	40.4	34.4	30.7
ANC (K/ul)	0.07	0.1	0.05	0.1	0.13	0.42	0.85	0.41	0.67	0.61	1.84	3.49	4.8	6.71	6.24	5.62	5.35

Fig. 1. Serial change of WBC, ANC, Hb, and platelets after ATRA, rhG-CSF, and erythropoietin therapy. RhG-CSF caused a mobilization effect of WBC. ANC began to increase after rhG-CSF was discontinued.

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Symptomatic Porphyria Cutanea Tarda and B-Immunoblastic Lymphoma: Is There an Association?

To the Editor: Prophyria cutanea tarda (PCT) is the most common form of hepatic porphyria. It has been associated with malignancies including hepatocellular carcinoma, leukemia, multiple myeloma, and lymphoma [1–5]. The initial possible association between PCT and lymphoma was described by Rayhanzadeh et al. [1]. Subsequent case reports further suggested that an association may be present [2–5].

We report a case of a 54-year-old businesswoman who presented with several months of intermittent, vesicular, tender skin lesions on the dorsum of both hands and occasionally on the face, ulcerating and resolving spontaneously. She also had a history of skin fragility and pruritus of both hands. She had no previous history of significant alcohol use, iron supplementation, hepatitis C infection, or family history of PCT. She had been started on estrogen replacement postoophorectomy 2 years earlier.

Her examination showed multiple, slightly raised erythematous lesions on the dorsum of both hands, and a slightly tender 3 × 4 cm fixed left axillary mass. Her liver enzymes were normal, and a 24-hr urine study for porphyrin showed elevated uroporphyrin (1,225 μg/day; normal, 0–50 μg/day) and coproporphyrin (335 μg/day; normal, 80–250 μg/day). Infection with hepatitis C virus and iron overload conditions were excluded. A biopsy of the left axillary node confirmed B-immunoblastic lymphoma. Further staging workup resulted in the diagnosis of stage II B-immunoblastic lymphoma. Estrogen replacement was stopped, and the patient was treated with six cycles of chemotherapy (cyclophosphamide, doxorubicin, vincristine, and prednisone (CHOP)) with complete remission or her lymphoma. Her biochemical abnormalities for PCT remained elevated for 12 months posttreatment (urine uroporphyrin ranged from 1,445–2,775 μg/24 hr, and urine coproporphyrin ranged from 361–694 μg/24 hr).

This patient's concomitant diagnosis of PCT with lymphoma again raises the possibility of an association between these two conditions. In all previously described cases, the clinical course of PCT in lymphoma patients has varied widely during treatment. None of these patients survived long enough to comment further on the course of their PCT [1–5]. In all these cases, including ours, the criteria to establish an association between these two conditions have not been fulfilled. Furthermore, we note that our patient, despite an excellent clinical response of her lymphoma to treatment, continued to have biochemical activity of PCT.

In conclusion, although the association between lymphoma and PCT is still possible, the current data, including our patient's course, do not support it. More patients with these entities are needed to be followed clinically to draw any solid conclusions.

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